

Temporal Context Cues in Human Fear Conditioning: Unreinforced Conditional Stimuli
can Segment Learning into Distinct Temporal Contexts and Drive Fear Responding

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Abstract

In associative learning, if stimulus A is presented in the same temporal context as the conditional stimulus (CS) - outcome association (but not in a way that allows an A–CS association to form) it becomes a temporal context cue, acquiring the ability to activate this context and retrieve the CS-outcome association. We examined whether a CS- presented during acquisition or extinction that predicted the absence of the unconditional stimulus (US) could act as a temporal context cue, reducing or enhancing responding, in differential fear conditioning. Two groups received acquisition (CSx-US, CSa –noUS) in phase 1 and extinction (CSx-noUS; CSe-noUS) in phase 2 (AE groups), and two groups received extinction in phase 1 and acquisition in phase 2 (EA groups). After a delay, participants were presented with either CSa (AEa and EAa groups) or CSe (AEe and EAe groups). Responding to CSx was enhanced after presentation of CSa but reduced after presentation of CSe, suggesting that training was segmented into two learning episodes and that the unreinforced CS present during an episode retrieved the CSx-US or CSx-noUS association. These findings suggest that temporal context cues may enhance or reduce fear responding, providing an exciting new avenue for relapse prevention research.

Key words: Fear conditioning, episodic memory, reinstatement, fear relapse, electrodermal responding

Anxiety researchers and clinicians have a common problem – anxiety disorders are particularly susceptible to relapse. Although treatments are efficacious in the short-term, between one and two thirds of successfully treated patients will relapse within eight years (Craske, 1999). Understanding what triggers relapse, how treatments can be made more robust against these triggers, and what aspects of fear acquisition make relapse more likely to occur is crucial. We have moved well past the assumption that extinction, or exposure, simply erases the original fear memory. Bouton's theory of relapse revolutionised the field – treatment does not erase the original fear learning but instead creates a context specific inhibitory learning – in this place, at this time, the original fear learning does not hold (Bouton, 2002). Context, however, is a complex concept – while a change in physical context is relatively concrete, a change in temporal context is not. Time is always moving and changing, being segmented into distinct temporal episodes. It is not clear what is encoded in these temporal episodes and whether stimuli that are present during a particular temporal episode can promote or reduce fear relapse.

Differential fear conditioning provides a reliable paradigm to study fear acquisition, extinction, and relapse (Vervliet, Craske, & Hermans, 2013). During differential fear acquisition, one neutral conditional stimulus (CS+; e.g., a picture of a circle) is paired with an aversive unconditional stimulus (US; e.g., an electro-tactile stimulus), while, a second neutral stimulus (CS-; e.g., a picture of a square) is presented alone. Throughout acquisition, differential physiological responding develops, such that the CS+ elicits larger physiological responses than the CS-. During fear extinction, the CS+ and the CS- are both presented alone, in the absence of the US, and the differential responding acquired throughout acquisition gradually reduces (Lipp, 2006). Extinction training creates an inhibitory association (CS+-noUS) which suppresses the excitatory fear association (CS+-US). After extinction, the CS+ becomes ambiguous and context

can be used to disambiguate it, i.e., context cues determine whether conditional fear returns (Bouton, 2002). In the laboratory, return of fear after successful extinction can be induced via three manipulations: unpaired presentations of the US alone (reinstatement), a context change after extinction (renewal), and testing after a delay (spontaneous recovery). Bouton suggests that reinstatement and spontaneous recovery could be regarded as special cases of renewal. Spontaneous recovery may occur because the CS+ is presented in a different temporal context and reinstatement may occur because presenting the US alone activates the CS+-US memory which triggers the acquisition context (Bouton, 2002; for a comprehensive review of return of fear mechanisms see Vervliet et al., 2013).

As context is critical in disambiguating the CS+ when two competing associations are present, researchers have tried presenting cues from extinction to increase the likelihood that participants will retrieve the inhibitory association. Presenting a cue (e.g., an ‘&’ symbol) on the screen during extinction (Dibbets, Havermans, & Arntz, 2008; Dibbets & Maes, 2011) or pairing another stimulus with the CS+ during extinction (Vansteenwegen, Vervliet, Hermans, Beckers, Baeyens, & Eelen, 2006) have been shown to attenuate renewal when these cues are also present during test. Retrieval cues have also been examined in clinical studies. Shin and Newman (2018) showed that using retrieval cues from exposure therapy (e.g. a puffer ball and a peppermint diffuser) could attenuate spontaneous recovery when participants had access to the cue at test. Culver, Stoyanova, and Craske (2011) and Dibbets, Moor, and Voncken (2013) examined the use of retrieval cues during exposure therapy, but both found that they did not attenuate fear renewal. Retrieval cues¹ are trained in a way that permits the formation of a direct association

¹ In this paper we refer to retrieval cues as cues that have been trained in a way that permits the formation of a direct association between the cue and the CS+ and to temporal context reminder cues (or just reminder cues) as cues that are trained in a way that does not allow the formation of a direct association between the cue and the CS+.

between the cue and the CS+. This direct association is problematic because the cues may function as conditioned inhibitors, protecting the CS+ from undergoing extinction learning altogether (Lovibond, Davis, & O'Flaherty, 2000; Rescorla & Wagner, 1972) and return of fear may occur when the CS+ is presented without them.

Evidence from the memory literature suggests that the content of temporal episodes can be triggered by stimuli that were present during the episode but not in a way that permitted the formation of a direct association with any other event from that episode. The absence of a direct association among events means that the stimulus used to retrieve a temporal episode will not act as a conditioned inhibitor or an occasion setter for other associations. Howard and Kahana's (2002) temporal context model proposes that during training, stimuli become associated with the current state of a gradually changing representation of the temporal context. This temporal context also enters into an association with the training stimuli such that subsequent presentation of a training stimulus can activate the temporal context. Based on this theory, Matute, Lipp, Vadillo, and Humphreys (2011) examined whether temporal context cues could enhance or reduce responding acquired during a causal learning task. During phase 1 of their experiments, stimulus X was repeatedly paired with outcome 1 and stimulus A with outcome 2; whereas in phase 2, stimulus X was repeatedly paired with outcome 2 and stimulus E with outcome 1. This training should render stimulus X ambiguous in a delayed test and, according to associative learning theories, should not permit a direct association between stimulus X and A, or stimulus X and E. Interestingly, the meaning of stimulus X was disambiguated when participants were presented with A followed by outcome 2 or E followed by outcome 1 prior to test. Participants behaved as if stimulus X was followed by outcome 1 after the A-outcome 2 pairing and by outcome 2 after the E-outcome 1 pairing. These results are interesting as they suggest that

temporal contexts can be defined not only by the mere passage of time but also by discrete stimuli present during a learning episode.

This result is especially relevant to anxiety researchers and clinicians as it would suggest that renewal can occur under a broader set of conditions than previously thought. Changes in context could be cued not only by physical changes in external and internal environments or by the passage of time, but also by any other stimulus that had been present during acquisition or extinction training. This would suggest that the presentation of stimuli that are associated with the treatment context could reduce relapse without becoming conditioned inhibitors and interfering with the extinction or exposure treatment. It would also suggest that stimuli that were present when the fear was acquired could activate the temporal context of acquisition and lead to relapse. Understanding whether temporal context cues can influence the retrieval of previously acquired or extinguished fear learning could aid the development of anxiety treatments and help us to understand, and prevent, instances of relapse. We examined whether the findings of Matute et al. (2011) in a causal learning task would transfer to fear conditioning involving the measurement of physiological fear responses. Using a differential fear conditioning design, half of the participants received acquisition training in phase 1, in which stimulus X was followed by an aversive US and stimulus A was presented alone, and extinction training in phase 2 in which both stimulus X and E were presented alone (AE groups). The other half of the participants received the same training but the order of the phases was reversed (extinction then acquisition; EA groups²). After a delay phase, participants were presented with one temporal context

² The extinction phase in this group could also be conceptualized as habituation training rather than extinction. The goal was to create two training phases that made the meaning of CSx ambiguous (i.e. trained competing CSx-US and CSx-noUS associations) and to ensure that the results held regardless of the order in which the associations were acquired. Therefore, we included this as a factor in the experiment. For simplicity, we have called the phases 'acquisition' and 'extinction' but they could also be referred to as the 'CSx-US' and CSx-noUS' phases.

reminder trial of either stimulus A (AEa and EAa groups) or stimulus E (AEe and EAe) and physiological responding to X was tested. We hypothesized that presentation of the acquisition reminder cue would enhance responding to X, and presentation of the extinction reminder cue would reduce responding to X in comparison to the last presentation of X during phase 2, regardless of whether phase 2 was acquisition or extinction.

Method

Participants. Sixty-eight undergraduate students aged between 17 and 41 years ($M = 21.29$, $SD = 4.20$ years) volunteered participation in exchange for course credit (46) or AU\$10 (22). Participants provided informed consent and were randomly assigned to one of four groups (AEa, AEe, EAa, EAe; $n = 19, 16, 16, 17$, respectively). The experimental procedure was approved by the local ethical review committee (approval number 2011001267). Data from 2 additional participants were lost due to a computer error.

Apparatus/Stimuli. The conditional stimuli were pictures of geometric shapes (black outlines on a white background; circle, square, diamond, upward pointing triangle, downward pointing triangle). Conditional stimuli were displayed on a 17-inch colour CRT screen and took up an area of approximately $6.5\text{cm} \times 6.5\text{cm}$. Three shapes were used as conditional stimuli during the main experiment and two as conditional stimuli during the delay phase. The CS+ from the main training phase will be referred to from now on as the CSx. CSx+ denotes that the CSx is reinforced, CSx- denotes that the CSx is not reinforced, and CSx is used when referring to the stimulus per se. The CS- from acquisition and the CS- from extinction will be referred to as CSa- and CSe-, respectively. Two different shapes were used as the CS+ and CS- during the delay and are referred to as CSg+ and CSh-, respectively. The square, circle, and diamond were used as CSx, CSa-, and CSe- and the two triangles were used as the unrelated CSg+ and CSh- stimuli in

the delay phase. The allocation of image to stimulus condition was counterbalanced across participants. A 200ms electro-tactile stimulus was used as the unconditional stimulus (US) and was generated by a Grass SD9 Stimulator pulsed at 50 Hz and presented via a concentric electrode filled with two saline soaked sponges and attached to the inside of the participants' preferred forearm. Stimulus presentation was controlled with DMDX software (Forster & Forster, 2003). Electrodermal activity was monitored with two 8 mm diameter Ag/AgCl electrodes filled with an isotonic electrolyte (TD-246 skin conductance paste) and attached to the thenar and hypothenar prominences of the participants' non-preferred hand. Respiration was monitored with an elasticized chest gauge. Physiological responses and signal markers were recorded with a Biopac MP150 system at a sampling frequency of 1000 Hz.

Procedure. Upon arrival at the laboratory, participants were informed about the general procedure and provided informed consent. They were seated in a recording room, adjacent to the control room, in front of the monitor and the measurement devices were attached. The experiment commenced with a shock work-up procedure during which the intensity of the electrotactile stimulus was set individually to be 'unpleasant, but not painful'. This was followed by a three minute baseline recording to determine participants' level of electrodermal responsiveness. After the baseline recording, all participants were presented with a sequence of two training phases (acquisition and extinction), a delay phase, a temporal context reminder trial and a test trial without interruption. The experimental groups differed in the sequence of acquisition and extinction training (groups AEa and AEe received acquisition first; groups EAa and EAe received extinction first) and whether CSa- or CSe- was presented on the temporal context reminder trial (groups AEa, EAa were presented with CSa-, groups AEe and EAe were presented with CSe-). Table 1 summarizes the training sequences for the different groups.

Acquisition/extinction comprised eight presentations each of CSx+ and CSa-/ CSx- and CSe-, respectively. The offset of the 6 s CSx coincided with the onset of the US (delay conditioning; interstimulus interval of 6 s) during acquisition (CSx+) whereas no US was presented during extinction (CSx-). During the delay phase, each participant was presented with four trials of CSg+ followed by the US and CSh- presented alone, respectively. The delay phase separated the training phases from the temporal context reminder trial³. During acquisition, extinction, and delay, the conditional stimuli were presented in a pseudo random order with no more than two consecutive trials being the same. Serial positions of CSx+/CSa-, CSx-/CSe-, and CSg+/CSh- were counterbalanced across participants. Trials were separated by intertrial intervals of 11, 13, or 15s, scheduled at random. The delay phase was followed by the temporal context reminder trial, a single presentation of CSa- or CSe-, and the test trial, a single presentation of CSx- without the US. All trials were run without interruption and transition from one phase to the next was not signaled to the participants. After the presentation of the test trial, the experimenter entered the participants' room and removed the measurement devices. Participants were then asked to rate the pleasantness of the five images presented during the experiment and of the US. They were also asked to indicate which of the three images were used as CSx, CSa-, and CSe-, and which of the images used as CSg+ and CSh- had been followed by shock. After this, participants were debriefed and thanked.

Coding and Statistical Analyses. Electrodermal responses were scored in three latency windows as recommended by Prokasy and Kumpfer (1973) and Luck and Lipp (2016). First interval responding was scored as the largest response (magnitude from response onset to response peak) starting within 1-4s of CS onset. Second interval responding was scored as the

³ The results of the delay phase have not been reported as it served as a filler task and was not relevant to the research question. The results, however, are available on request from the corresponding author.

largest response starting within 4-7s of CS onset. Responses to the US were scored during acquisition as the largest response starting within 7-10s of CS onset (1-4s from US onset). Both first and second interval responding are sensitive to fear learning, but first interval responses are more sensitive to orienting processes and second interval responses are more sensitive to anticipatory processes (Luck & Lipp, 2016). Respiration traces were examined as a control measure to identify trials where electrodermal responding was contaminated by deep breaths or excessive movement, however, no such trials were identified and therefore no responses were removed. To reduce the positive skew of the distribution, electrodermal responses were square root transformed (Dawson, Schell, & Filion, 2007) and then range corrected to give each participant an even weight in the analysis. The largest response displayed by a participant was used as the reference for the range correction. All analyses were conducted with IBM SPSS Statistics 24 software and an alpha cut-off of .05. *F* values (Phillai's Trace) are reported for all analyses.

Results

Preliminary Analyses

The means and standard deviations for variables assessed in the preliminary analyses are presented in Table 2. Pearson's chi square tests confirmed that the ratio of males and females, $\chi^2(3) = 5.61, p = .132$, and of contingency passes and fails⁴, $\chi^2(3) = 3.95, p = .266$, did not differ across the groups. Unconditional electrodermal responding during acquisition was subjected to a 2 Phase Sequence (acquisition first, extinction first) \times 2 Reminder Cue (acquisition, extinction) \times 4 Block (1, 2, 3, 4) mixed-model ANOVA. Unconditional electrodermal responses declined

⁴ The analyses were also run excluding the participants who did not pass the contingency check and the pattern of results did not change, therefore results from the entire sample have been reported.

across blocks, $F(3, 62) = 16.65, p < .001, \eta^2 = .446$. A main effect of phase sequence, $F(1, 64) = 4.84, p = .031, \eta^2 = .070$, was moderated by a Phase Sequence \times Reminder Cue interaction, $F(1, 64) = 4.68, p = .034, \eta^2 = .068$. In the groups who received acquisition training first, there was no difference in unconditional responding between groups receiving the acquisition cue (AEa) and the extinction cue (AEe), $F(1, 64) < 0.01, p = .979, \eta^2 < .001$. In the groups who received extinction training first, the group receiving the extinction cue (EAe) showed larger unconditional responses than the group who received the acquisition cue (EAa), $F(1, 64) = 9.27, p = .003, \eta^2 = .127$. The remaining effects did not reach significance, $F < 2.00, p > .123, \eta^2 < .089$.

A series of 2 Phase Sequence (acquisition first, extinction first) \times 2 Reminder Cue (acquisition, extinction) between-participant ANOVAs were run on the remaining preliminary variables. The groups did not differ in CSx, CSa-, or CSe- valence ratings⁵. The groups presented with the acquisition cue evaluated the US as more unpleasant than the groups presented with the extinction cue, $F(1, 65) = 5.62, p = .021, \eta^2 = .080$. The groups who completed extinction training first displayed more spontaneous electrodermal responses in baseline than the groups who underwent acquisition training first, $F(1, 42) = 4.50, p = .040, \eta^2 = .097$. The participants who received the extinction cue were older than the participants who received the acquisition cue, $F(1, 64) = 8.04, p = .006, \eta^2 = .112$. All other differences did not reach significance, $F < 2.07, p > .158, \eta^2 < .048$. Although there were some differences across groups in overall responsiveness and evaluation of the US, these baseline differences would suggest elevated

⁵ The groups did not differ in how they evaluated the different stimuli, but all participants evaluated the CSx as less pleasant than the CSa, $p < .001$, and CSe, $p < .001$. Evaluations of CSa- and CSe- did not differ, $p = .128$.

responses in the groups presented with CSe- as temporal reminder cue and thus run contrary to the predicted pattern of results.

Conditioning Training

The first and second interval electrodermal responses from Phase 1 and 2 were subjected to separate 2 CS (CSx, CSa-/CSe-) \times 2 Phase Sequence (acquisition first, extinction first) \times 2 Reminder Cue (acquisition, extinction) \times 4 Block (1, 2, 3, 4) mixed model ANOVAs and are displayed in Figures 1, and 2, respectively.⁶

Phase 1 - First Interval Responding. Main effects of CS, $F(1, 64) = 46.62, p < .001, \eta^2 = .421$, and block, $F(3, 62) = 18.71, p < .001, \eta^2 = .475$, were moderated by CS \times Phase Sequence, $F(1, 64) = 56.01, p < .001, \eta^2 = .467$, and CS \times Phase Sequence \times Block interactions, $F(3, 62) = 2.89, p = .042, \eta^2 = .123$. Follow-up analyses revealed that in the groups undergoing acquisition first, responses to CSx+ were larger than responses to CSa- during blocks one, $F(1, 64) = 16.79, p < .001, \eta^2 = .208$, two, $F(1, 64) = 62.11, p < .001, \eta^2 = .492$, three, $F(1, 64) = 44.34, p < .001, \eta^2 = .409$, and four, $F(1, 64) = 11.30, p = .001, \eta^2 = .150$. In the groups undergoing extinction first, responses to CSx- did not differ from responses to CSe- during blocks one, $F(1, 64) = 0.39, p = .534, \eta^2 = .006$, two, $F(1, 64) = 0.01, p = .930, \eta^2 < .001$, three, $F(1, 64) = 2.39, p = .127, \eta^2 = .036$, or four, $F(1, 64) = 0.03, p = .875, \eta^2 < .001$.⁷ The remaining main effects and interactions did not reach significance, $F < 2.72, p > .052, \eta^2 < .117$.⁸

⁶ The results analysed based on conditioning phase are reported in the supplement.

⁷ The three way interaction reflects changes in the size of the differential response across blocks.

⁸ The effect closest to significance is the Block \times Phase Sequence interaction which is moderated by a higher order interaction in this analysis.

Phase 1 - Second Interval Responding. A main effect of CS, $F(1, 64) = 20.67, p < .001$, $\eta^2 = .244$, and a CS \times Phase Sequence interaction, $F(1, 64) = 5.17, p = .026, \eta^2 = .075$, were moderated by a CS \times Phase Sequence \times Block interaction, $F(3, 62) = 3.45, p = .022, \eta^2 = .143$. Follow-up analyses revealed that in the groups undergoing acquisition first, responses to CSx+ and CSa- did not differ during block one, $F(1, 64) = 0.70, p = .406, \eta^2 = .011$, however during blocks two, $F(1, 64) = 7.36, p = .009, \eta^2 = .103$, three, $F(1, 64) = 15.85, p < .001, \eta^2 = .198$, and four, $F(1, 64) = 12.18, p = .001, \eta^2 = .160$, responding to CSx+ exceeded responding to CSa-. In the groups undergoing extinction first, responses to CSx- did not differ from responses to CSe- during blocks one, $F(1, 64) = 0.41, p = .526, \eta^2 = .006$, three, $F(1, 64) = 0.49, p = .485, \eta^2 = .008$, or four, $F(1, 64) = 0.46, p = .500, \eta^2 = .007$. Unexpectedly, during block two, responding to CSx- exceeded responding to CSe-, $F(1, 64) = 7.41, p = .008, \eta^2 = .104$. The remaining main effects and interactions did not reach significance, $F < 2.48, p > .070, \eta^2 < .108$.

Phase 2 - First Interval Responding. Main effects of CS, $F(1, 64) = 8.75, p = .004, \eta^2 = .120$, and block, $F(3, 62) = 7.51, p < .001, \eta^2 = .267$, were moderated by a CS \times Block interaction, $F(3, 62) = 9.29, p < .001, \eta^2 = .310$, and a marginal CS \times Phase Sequence interaction, $F(1, 64) = 3.46, p = .067, \eta^2 = .051$. Follow-up analyses of the CS \times Block interaction revealed that, across phase sequence groups, responding to the CSx was marginally larger than to CSa-/CSe- during block one, $F(1, 64) = 3.81, p = .055, \eta^2 = .056$. During block two, there was no difference in responding between the CSx and the CSa-/CSe-, $F(1, 64) = 1.71, p = .196, \eta^2 = .026$, and during blocks three, $F(1, 64) = 15.56, p < .001, \eta^2 = .196$, and four, $F(1, 64) = 18.20, p < .001, \eta^2 = .221$, responding to CSx was larger than to the CSa-/CSe-. Follow-up of the marginal CS \times Phase Sequence interaction confirmed that responding to CSx- and CSe- did not differ in the groups undergoing extinction training (AE groups), $F(1, 64) =$

0.62, $p = .435$, $\eta^2 = .010$, but that responding to CSx+ exceeded responding to CSa- in the groups undergoing acquisition training (EA groups), $F(1, 64) = 11.31$, $p = .001$, $\eta^2 = .150$. The remaining main effects and interactions did not reach significance, all $F < 3.02$, $p > .087$, $\eta^2 < .046$.

Phase 2 - Second Interval Responding. A main effect of CS, $F(1, 64) = 13.70$, $p < .001$, $\eta^2 = .176$, a CS \times Block interaction, $F(3, 62) = 2.79$, $p = .048$, $\eta^2 = .119$, a Reminder Cue \times Block interaction, $F(3, 62) = 3.11$, $p = .033$, $\eta^2 = .131$, and a CS \times Phase Sequence interaction, $F(1, 64) = 7.47$, $p = .008$, $\eta^2 = .105$, were detected. Follow-up analyses of the CS \times Block interaction revealed that, across phase sequence groups, responding between the CSx and CSa-/e- did not differ during block one, $F(1, 64) = 0.18$, $p = .670$, $\eta^2 = .003$. During blocks two, $F(1, 64) = 5.21$, $p = .026$, $\eta^2 = .075$, three, $F(1, 64) = 8.11$, $p = .006$, $\eta^2 = .113$, and four, $F(1, 64) = 13.83$, $p < .001$, $\eta^2 = .178$, however, responding to the CSx was larger than to the CSa-/CSe-. The CS \times Phase Sequence interaction confirmed that responding to CSx- and CSe- did not differ in the groups undergoing extinction training (AE groups), $F(1, 64) = 0.48$, $p = .490$, $\eta^2 = .007$, but that responding to CSx+ exceeded responding to CSa- in groups undergoing acquisition training (EA groups), $F(1, 64) = 20.17$, $p < .001$, $\eta^2 = .240$. The Reminder Cue \times Block interaction revealed that overall responding was larger in the groups who would receive the acquisition cue during block one, $F(1, 64) = 4.78$, $p = .033$, $\eta^2 = .069$, but did not differ between the temporal context reminder cue groups during blocks two, $F(1, 64) = 0.10$, $p = .753$, $\eta^2 = .002$, three, $F(1, 64) = 0.52$, $p = .472$, $\eta^2 = .008$, or four, $F(1, 64) = 0.69$, $p = .408$, $\eta^2 = .011$.

The remaining main effects and interactions did not reach significance, all $F < 3.65$, $p > .060$, $\eta^2 < .055$ ⁹.

Test Phase

The influence of the temporal context reminder trial¹⁰ on first and second interval responding to the CSx was analysed with separate 2 Phase Sequence (acquisition first, extinction first) \times 2 Reminder Cue (acquisition, extinction) \times 2 Trial (last CSx trial of phase 2, CSx test trial) mixed-model ANOVAs and are presented in Figures 3, and 4, respectively.

First Interval Responding. No significant effects were detected, all $F < 2.80$, $p > .099$, $\eta^2 < .043$.

Second Interval Responding. A Trial \times Reminder Cue interaction, $F(1, 64) = 7.69$, $p = .007$, $\eta^2 = .107$, revealed that presentation of the extinction reminder cue decreased responding to CSx from the last trial of phase 2 to the test trial, $F(1, 64) = 4.92$, $p = .030$, $\eta^2 = .071$.

Presentation of the acquisition cue marginally increased responding to CSx from the last trial of phase 2 to the test trial, $F(1, 64) = 2.89$, $p = .094$, $\eta^2 = .043$. The effect of the acquisition cue is likely dampened by ceiling effects due to the fact that CSx during the last trial of phase 2 in group EAa is a valid predictor of the US which would suggest maximum responding to the CSx during the last trial of phase 2. Analysis of the influence of presenting CSa in the AEa group

⁹ The effect closest to significance is the main effect of phase sequence which is moderated by a higher order interaction in this analysis.

¹⁰ There were no differences between the groups in first interval, $F < 1.29$, $p > .261$, $\eta^2 < .020$, or second interval responding to the temporal context reminder cue, $F < 2.90$, $p > .093$, $\eta^2 < .044$. Mean responses to the temporal context reminder cue are reported in Table 2.

revealed that responding to CSx significantly increased from the last trial of phase 2 (extinction) to the test trial, $F(1, 64) = 5.28$, $p = .025$, $\eta^2 = .076$, confirming that the acquisition cue did increase responding to CSx. The remaining omnibus effects did not reach significance, all $F < 3.77$, $p > .056$, $\eta^2 < .056$ ¹¹.

Discussion

The current study examined whether stimuli presented during fear acquisition and extinction, but not in a way that permitted the formation of a direct association with the CS+ or the US, could enhance or reduce fear responding, respectively. After completing acquisition and extinction training, participants were presented with the CS- from acquisition (CSa-) or extinction (CSe-) and then responding to the CS+ (CSx) was examined. As predicted, the presentation of CSe- during reminder reduced second interval electrodermal responding, while, presentation of CSa- during reminder increased second interval electrodermal responding in the group that completed extinction training in phase 2. The findings replicate the results of Matute et al. (2011) and extend them to a fear conditioning paradigm in which physiological responses are measured instead of behavioral responses or self-report. While, the temporal context cues influenced second interval electrodermal responding in the predicted direction, they did not influence first interval electrodermal responding; however, dissociations between first and second interval responding are common (see Luck & Lipp, 2016) and in this instance, likely occur because temporal context cues influence responding mostly via changes in anticipatory processes. The current findings are both exciting and concerning – they suggest that a temporal

¹¹ The effect closest to significance is the Phase Sequence \times Trial interaction which revealed that during the last trial of phase 2, the groups who completed acquisition in phase 2 (EA groups) had higher CSx responses than the groups who completed extinction in phase 2 (AE groups), $F(1, 64) = 5.98$, $p = .017$, $\eta^2 = .086$. Responses to CSx did not differ between the phase sequence groups during the test trial, $F(1, 64) = 0.20$, $p = .657$, $\eta^2 = .003$.

context cue presented during extinction can be used to reduce fear responses. This cue should be unlikely to act as a conditioned inhibitor or protect the CS+ from undergoing extinction learning due to the absence of a direct association between the cue and the CS+. On the other hand, however, they suggest that the conditions under which relapse can be triggered are broader than previously thought. Any stimulus present during fear acquisition, even a stimulus that predicted the absence of the US and could be conceptualized as a 'safety signal', could trigger the return of fear.

In the current study, the trial phases were presented without interruption and therefore the only possible contextual triggers were the stimuli presented during the same learning episode (i.e. CSa and CSx; CSe and CSx; CSg and CSh). Thus, the present findings suggest that participants segmented the experimental trial sequence into discrete learning episodes and that stimuli presented during the different learning episodes acquired the ability to activate the respective memory episode and retrieve the CSx-US or CSx-noUS association. This is in accordance with the temporal context model proposed by Howard and Kahana (2002) which suggests that the stimuli present during training become associated with the gradually changing representation of the temporal context and develop the ability to activate this temporal context memory.

Our findings are consistent with the principles of Bouton's theory of relapse but our manipulation seems to fall somewhere in-between renewal and reinstatement. Temporal renewal is typically defined by explicit changes in the passage of time, while reinstatement is suggested to retrieve the acquisition context by activating the CS+-US association. Similar to reinstatement, the presentation of a discrete stimulus from training was able to activate the temporal context of acquisition or extinction; however, unlike in reinstatement, this stimulus was not associated with

the US or the CS+. Similar to renewal, the change in context disambiguates the CS+, however, unlike renewal, the temporal context change is not induced by the mere passage of time but by discrete stimuli that were present during the different learning episodes.

One may argue that the recurrence or suppression of the conditional response observed during test was mediated by a retrieval cue (Brooks & Bouton, 1993) or by simple occasion setting (Holland, 1992). Both of these accounts would require a training procedure that allows an association to be formed between CSx and CSa during acquisition/CSx and CSe during extinction or between the CSa and the CSx-US association. These accounts, however, do not seem feasible as it is difficult to see how such an association could have formed bridging an inter-trial interval of 11-15s. Although, occasion setting has been shown with a gap between stimuli of 5 s (Hardwick & Lipp, 2000), it seems unlikely that a gap two to three times as long could be bridged after training that included only a very small number of trials where the CSx was preceded by the CSa/CSe and just as many trials where the CSx was preceded by the CSx or the CSa/CSe was followed by the CSa/CSe. Such accounts would also not be consistent with the findings of Matute et al. (2011; Experiment 4) in which the authors used a miscuing paradigm to examine whether presenting an outcome that participants were not expecting would lead to a temporal context memory update. In Phase 1, participants learned that X predicted outcome 1 and A predicted outcome 2, while, in Phase 2, participants learned that Y predicted outcome 1 and B predicted outcome 2. If A was presented with *outcome 1* during the temporal context reminder trial, participants responded to X as if it predicted *outcome 2*. Similarly, if B was presented with *outcome 1* during the temporal context reminder trial, participants responded to Y as if it predicted *outcome 2*. At no stage did the participants receive training in which X or Y predicted outcome 2 and therefore the findings cannot be explained by a simple associative

learning account but suggest that the X or Y cue retrieved the temporal context and that the unexpected outcome led to a memory update. Many memory researchers suggest that the purpose of retrieving an old temporal context is to guide present adaptive behavior (Matute et al., 2011).

Temporal context cues from extinction could be used to reduce relapse and should be preferable to retrieval cues as they will not act as a conditioned inhibitor that may prevent extinction/successful exposure therapy. Researchers and clinicians, however, should be cautious about relying on *any* cue to reduce fear responses in a clinical setting as these signals may not always be available. The results of Matute et al. (2011; Experiment 4) also lead to some additional exciting clinical possibilities. If the temporal context of the original fear learning is reactivated by presenting a cue that was present during acquisition, it may then also be possible to update the acquisition memory and reduce fear responding. Clinically, however, this is a challenging task – often clients will not remember the original fear acquisition itself, let alone other unrelated stimuli present in that temporal context. Moreover, the segmenting of a continuous experimental trial sequence that lasted approximately 20 minutes into temporal episodes based on the discrete stimuli present during the training phases, especially when these stimuli are the only contextual triggers available to use, may not be a proxy for real life where the external and internal environment is changing constantly, leading to temporal episodes that are longer and more variable. Thus, it is possible that temporal context cues are weak in comparison and easily overpowered by other physical and internal cues. Should this not be the case, however, temporal context cues could be a powerful tool for manipulating fear memories and thus offer a very interesting and exciting avenue for future research. Even if the original fear memory cannot be changed, temporal context cues offer the possibility to predict and further understand incidences of relapse. The current study provides a proof of concept – temporal

context cues can successfully reduce and enhance fear responding in human fear conditioning.

We hope that this proof of concept provides the starting point for researchers to develop interventions that offer more robust protection against relapse in the long-term.

Table 1

Experiential Procedure for the Different Groups

	Phase 1 Training	Phase 2 Training	Delay Phase	Temporal Context Reminder Cue	Test Stimulus
AEa	Acquisition	Extinction	4 CSg–US 4 CSh-	CSa-	CSx-
AEe	8 CSx+–US 8 CSa-	8 CSx- 8 CSe-		CSe-	
	EAa	Extinction		Acquisition	
EAe	8 CSx- 8 CSe-	8 CSx+–US 8 CSa-		CSe-	

Note. Participants were presented with acquisition (AEa, AEe) or extinction (EaA, EaE) training during Phase 1 and with CS_a– (AEa, EaA) or CSe– (AEe, EaE) as the temporal context reminder cue.

Table 2

Means and Standard Deviations for the Different Demographic Variables

	AEa	AEe	EAa	EAe
Age	20.42 (2.71)	23.19 (5.73)	19.44 (2.31)	22.24 (4.59)
Sex (male:female ratio)	9:10	3:13	5:11	4:13
Contingency Check (pass:fail ratio)	18:1	16:1	13:3	12:5
Spontaneous EDA	10.00 (12.71)	15.88 (12.82)	25.00 (14.09)	18.77 (13.02)
US Valence	2.84 (1.32)	2.29 (0.69)	2.56 (0.81)	2.06 (0.66)
CSx Valence	2.63 (1.01)	2.47 (1.13)	3.00 (1.21)	2.88 (1.76)
CSa- Valence	5.00 (1.49)	5.29 (1.57)	5.38 (1.46)	5.29 (1.65)
CSe- Valence	4.89 (1.52)	5.82 (1.47)	5.69 (1.20)	5.65 (1.66)
Unconditional responding	.58 (.21)	.48 (.11)	.58 (.23)	.67 (.16)
FIR to reminder cue	.27 (.28)	.17 (.27)	.20 (.23)	.15 (.25)
SIR to reminder cue	.13 (.24)	.08 (.14)	.20 (.29)	.07 (.17)

Note. Unconditional electrodermal responses are averaged across all blocks of acquisition.

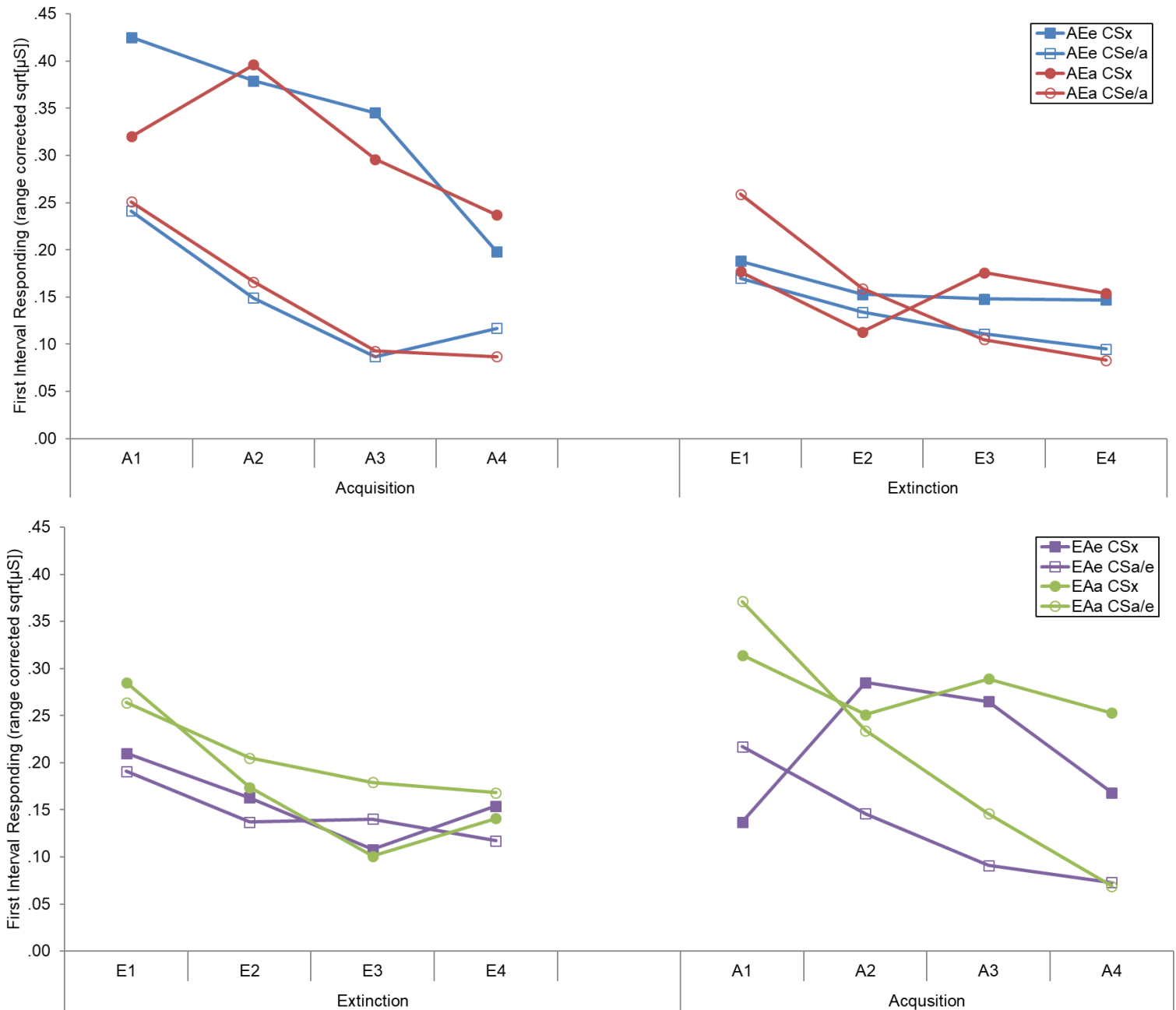


Figure 1. First interval electrodermal responding recorded during Phase 1 and Phase 2 for the different groups. During acquisition, the CSa- is presented and during extinction the CSe- is presented.

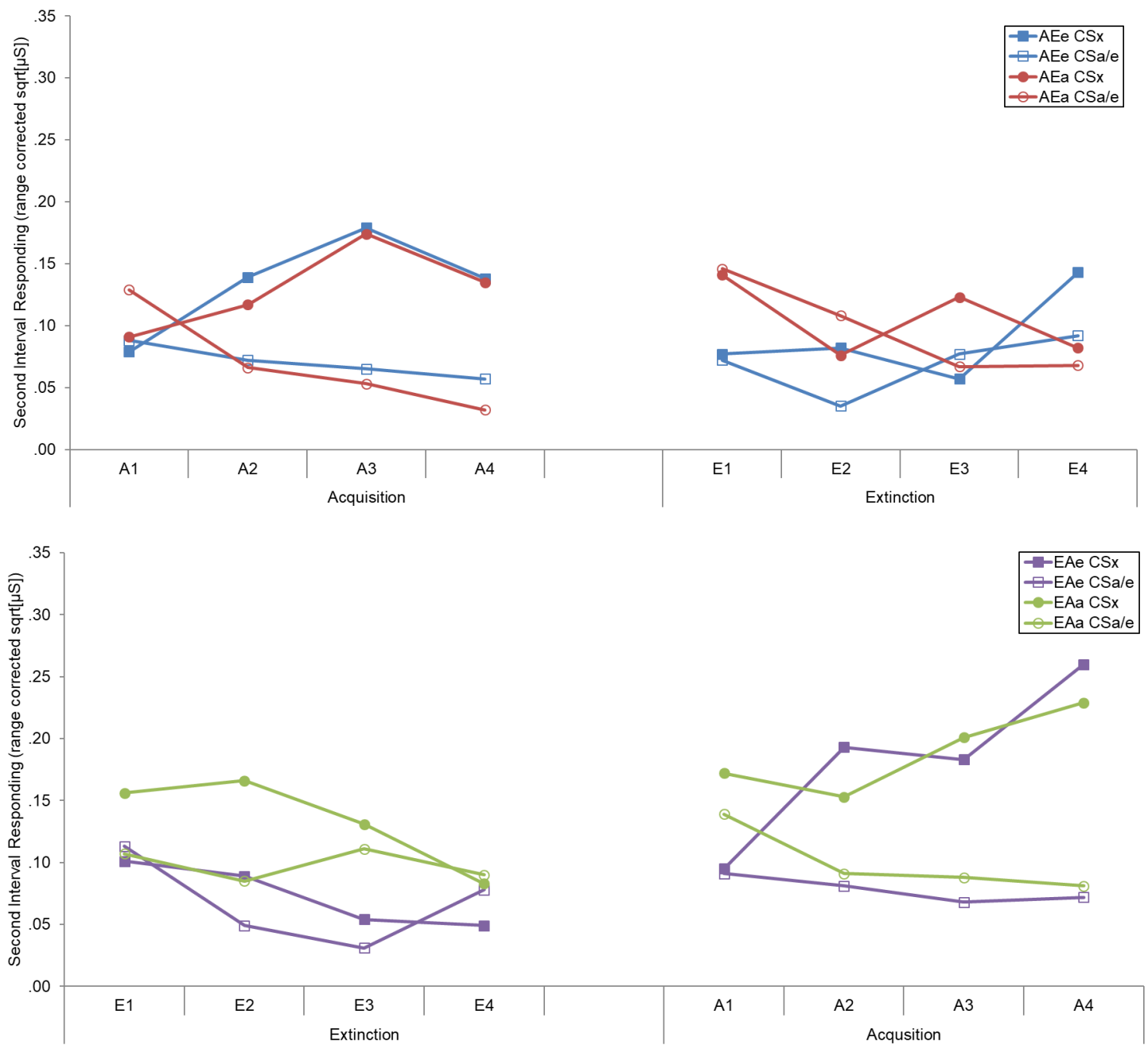


Figure 2. Second interval electrodermal responding recorded during Phase 1 and Phase 2 for the different groups. During acquisition, the CSa- is presented and during extinction the CSe- is presented.

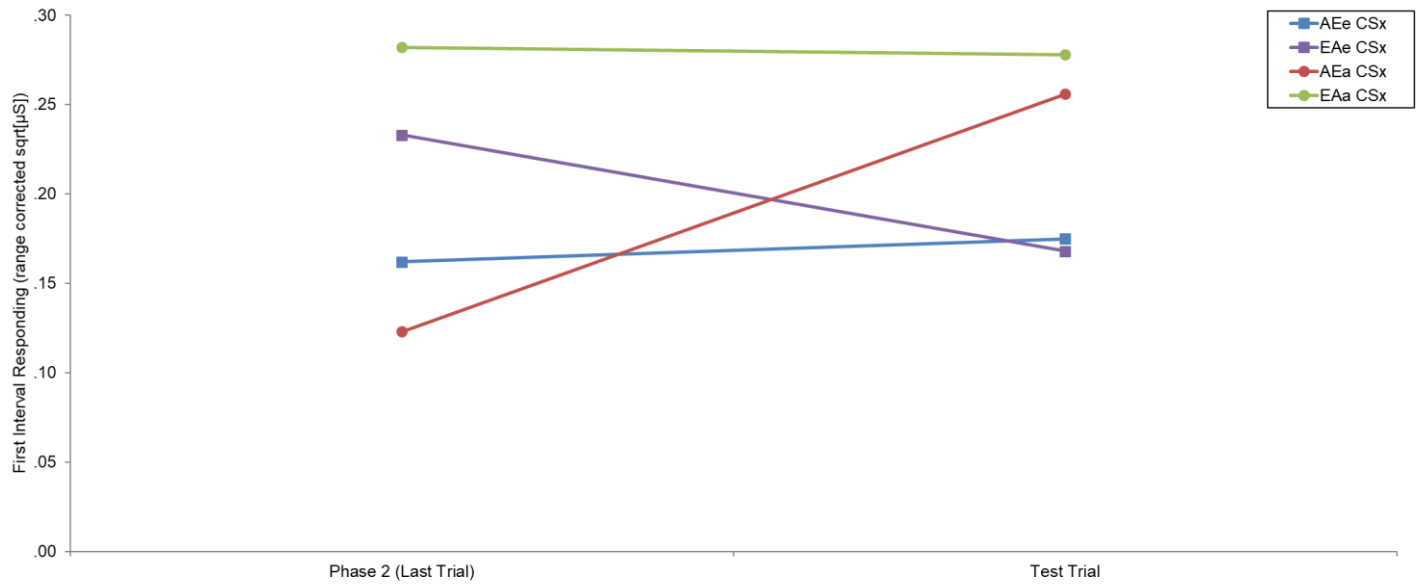


Figure 3. First interval electrodermal responding to CSx recorded during the last trial of Phase 2 and during test.

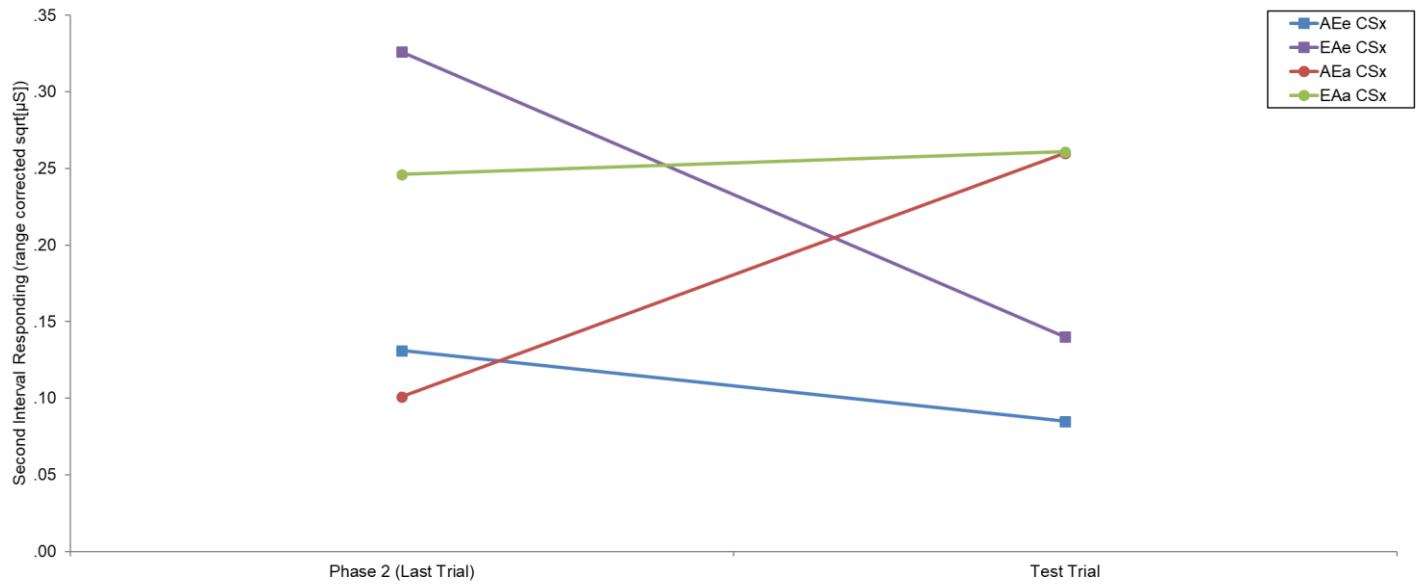


Figure 4. Second interval electrodermal responding to CSx recorded during the last trial of Phase 2 and during test.

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Supplementary Material

Acquisition

The first and second interval electrodermal responses from acquisition were subjected to separate 2 CS (CSx, CSa-) \times 2 Phase Sequence (acquisition first, extinction first) \times 2 Reminder Cue (acquisition, extinction) \times 4 Block (1, 2, 3, 4) mixed model ANOVAs and are displayed in Figures 1 and 2, respectively.

First Interval Responding. A main effect of CS, $F(1, 64) = 62.50, p < .001, \eta^2 = .494$, a main effect of block, $F(3, 62) = 16.91, p < .001, \eta^2 = .450$, a CS \times Phase Sequence interaction, $F(1, 64) = 9.54, p = .003, \eta^2 = .130$, and a CS \times Block interaction, $F(3, 62) = 6.04, p = .001, \eta^2 = .226$, were moderated by a CS \times Phase Sequence \times Block interaction, $F(3, 62) = 3.46, p = .022, \eta^2 = .143$. In the group undergoing acquisition first, responding to CSx+ exceeded responding to CSa- during blocks one, $F(1, 64) = 10.32, p = .002, \eta^2 = .139$, two, $F(1, 64) = 45.40, p < .001, \eta^2 = .415$, three, $F(1, 64) = 30.17, p < .001, \eta^2 = .320$, and four, $F(1, 64) = 11.16, p = .001, \eta^2 = .148$. In the groups undergoing acquisition after extinction, responding between CSx+ and CSa- did not differ during block one, $F(1, 64) = 2.90, p = .094, \eta^2 = .043$, but during blocks two, $F(1, 64) = 4.94, p = .030, \eta^2 = .072$, three, $F(1, 64) = 13.57, p < .001, \eta^2 = .175$, and four, $F(1, 64) = 15.57, p < .001, \eta^2 = .196$, responding to CSx+ was larger than to CSa-. The remaining main effects and interactions did not reach significance, $F < 2.37, p > .079, \eta^2 < .103$.

Second Interval Responding. A main effect of CS, $F(1, 64) = 25.63, p < .001, \eta^2 = .286$, was moderated by a CS \times Block interaction, $F(3, 62) = 5.42, p = .002, \eta^2 = .208$. During block one responding did not differ between CSx+ and CSa-, $F(1, 64) = 0.01, p = .918, \eta^2 < .001$.

.001, however, during blocks two, $F(1, 64) = 10.87, p = .002, \eta^2 = .145$, three, $F(1, 64) = 19.02, p < .001, \eta^2 = .229$, and four, $F(1, 64) = 21.86, p < .001, \eta^2 = .255$, responding to CSx+ exceeded responding to CSa-. The remaining main effects and interactions did not reach significance, $F < 2.51, p > .115, \eta^2 < .039$.

Extinction

The first and second interval electrodermal responses from extinction were subjected to separate 2 CS (CSx-, CSe-) \times 2 Phase Sequence (acquisition first, extinction first) \times 2 Reminder Cue (acquisition, extinction) \times 4 Block (1, 2, 3, 4) mixed model ANOVAs and are displayed in Figures 1 and 2, respectively.

First Interval Responding. A main effect of block, $F(3, 62) = 10.03, p < .001, \eta^2 = .327$, was moderated by a CS \times Phase Sequence \times Block interaction, $F(3, 62) = 4.00, p = .011, \eta^2 = .162$. In the groups undergoing extinction after acquisition, responding did not differ between CSx- and CSe- during blocks one, $F(1, 64) = 1.45, p = .232, \eta^2 = .022$, two, $F(1, 64) = 0.22, p = .641, \eta^2 = .003$, three, $F(1, 64) = 3.40, p = .070, \eta^2 = .050$, or four, $F(1, 64) = 3.57, p = .064, \eta^2 = .053$. In the groups undergoing extinction first, responding did not differ between CSx- and CSe- during blocks one, $F(1, 64) = 0.54, p = .467, \eta^2 = .008$, two, $F(1, 64) = 0.01, p = .931, \eta^2 < .001$, three, $F(1, 64) = 3.37, p = .071, \eta^2 = .050$, or four, $F(1, 64) = 0.03, p = .870, \eta^2 < .001$. Responding to CSx- and CSe- did not differ between the phase sequence groups during any block, $F < 1.95, p > .167, \eta^2 < .030$. The remaining main effects and interactions did not reach significance, $F < 2.01, p > .161, \eta^2 < .031$.

Second Interval Responding. A main effect of CS, $F(1, 64) = 4.82, p = .032, \eta^2 = .070$, revealed that responding to CSx- was larger than to CSe-. The remaining main effects and interactions did not reach significance, $F < 2.29, p > .087, \eta^2 < .100$.

Acquisition/Extinction Comparison

The first and second interval responding on the last block of acquisition and the last block of extinction were examined with separate 2 CS (CSx, CSa-/CSe-) \times 2 Phase Sequence (acquisition first, extinction first) \times 2 Reminder Cue (acquisition, extinction) \times 2 Phase (last block of acquisition, last block of extinction) mixed model ANOVAs.

First Interval Responding. A main effect of CS, $F(1, 64) = 19.16, p < .001, \eta^2 = .230$, was moderated by a CS \times Phase interaction, $F(1, 64) = 9.10, p = .004, \eta^2 = .124$. During the last block of acquisition, responding to CSx+ was larger than to CSa-, $F(1, 64) = 26.60, p < .001, \eta^2 = .294$, while, during the last block of extinction there was no difference in responding between the CSx- and the CSe-, $F(1, 64) = 2.06, p = .156, \eta^2 = .031$. The remaining main effects and interactions did not reach significance, $F < 2.65, p > .108, \eta^2 < .040$.

Second Interval Responding. A main effect of CS, $F(1, 64) = 18.55, p < .001, \eta^2 = .225$, and a main effect of phase, $F(1, 64) = 6.65, p = .012, \eta^2 = .094$, were moderated by a CS \times Phase interaction, $F(1, 64) = 13.11, p = .001, \eta^2 = .170$, and a Phase Sequence \times Phase interaction, $F(1, 64) = 8.55, p = .005, \eta^2 = .118$. The CS \times Phase interaction revealed that during the last block of acquisition, responding to CSx+ was larger than to CSa-, $F(1, 64) = 21.86, p < .001, \eta^2 = .255$, while, during the last block of extinction there was no difference in responding between the CSx- and the CSe-, $F(1, 64) = 0.16, p = .692, \eta^2 = .002$. The Phase Sequence \times

Phase interaction revealed that overall responding was larger during the last block of acquisition, in the groups who completed extinction first, $F(1, 64) = 4.99, p = .029, \eta^2 = .072$, but overall responding did not differ between the phase sequence groups during the last block of extinction, $F(1, 64) = 0.65, p = .422, \eta^2 = .010$. Note: this is overall responding not conditional responding – the difference revealed during acquisition by the Phase Sequence \times Phase interaction likely just reflects that orienting responses had decreased in the group undergoing extinction before acquisition. The remaining main effects and interactions did not reach significance, $F < 3.47, p > .067, \eta^2 < .052$.

Test Phase

2 Phase Sequence (acquisition first, extinction first) \times 2 Reminder Cue (acquisition, extinction) \times 3 Trial (last CSx trial of acquisition, CSx trial of extinction, CSx test trial) mixed-model ANOVA and are displayed in Figures 3 and 4, respectively.

First Interval Responding. A main effect of trial, $F(2, 62) = 3.63, p = .032, \eta^2 = .105$, revealed that responding to CSx decreased from acquisition to extinction, $p = .010$, and marginally increased from extinction to test, $p = .058$. The remaining main effects and interactions did not reach significance, $F < 1.54, p > .223, \eta^2 < .048$.

Second Interval Responding. A main effect of trial, $F(2, 63) = 6.83, p = .002, \eta^2 = .178$, was moderated by a Phase Sequence \times Trial interaction, $F(2, 63) = 7.50, p = .001, \eta^2 = .192$, and a Reminder Cue \times Trial interaction, $F(2, 63) = 3.32, p = .042, \eta^2 = .095$. The Phase Sequence \times Trial interaction revealed that responding to CSx during the last trial of acquisition, was smaller in the groups who completed acquisition before extinction, $F(1, 64) = 9.55, p = .003, \eta^2 = .130$, while responding did not differ between the phase sequence groups after extinction,

$F(1, 64) = 1.01, p = .319, \eta p^2 = .016$, or at test, $F(1, 64) = 0.20, p = .657, \eta p^2 = .003$. The Reminder Cue \times Trial interaction revealed that while the reminder cue groups did not differ in responding to CSx during the last trial of acquisition, $F(1, 64) = 0.28, p = .599, \eta p^2 = .004$, and during the last trial of extinction, $F(1, 64) = 0.17, p = .685, \eta p^2 = .003$, responding to CSx was larger at test after viewing the acquisition reminder cue in comparison to the extinction cue, $F(1, 64) = 5.64, p = .021, \eta p^2 = .081$. A comparison of responding to CSx across trials confirmed that in the group who received the acquisition cue responding marginally increased from acquisition to test, $p = .083$, and significantly increased from extinction to test, $p = .001$. In the group receiving the extinction cue, responding to CSx marginally decreased from acquisition to test, $p = .084$, and did not change from extinction to test, $p = .513$. The remaining main effects and interactions did not reach significance, $F < 1.82, p > .182, \eta p^2 < .028$.

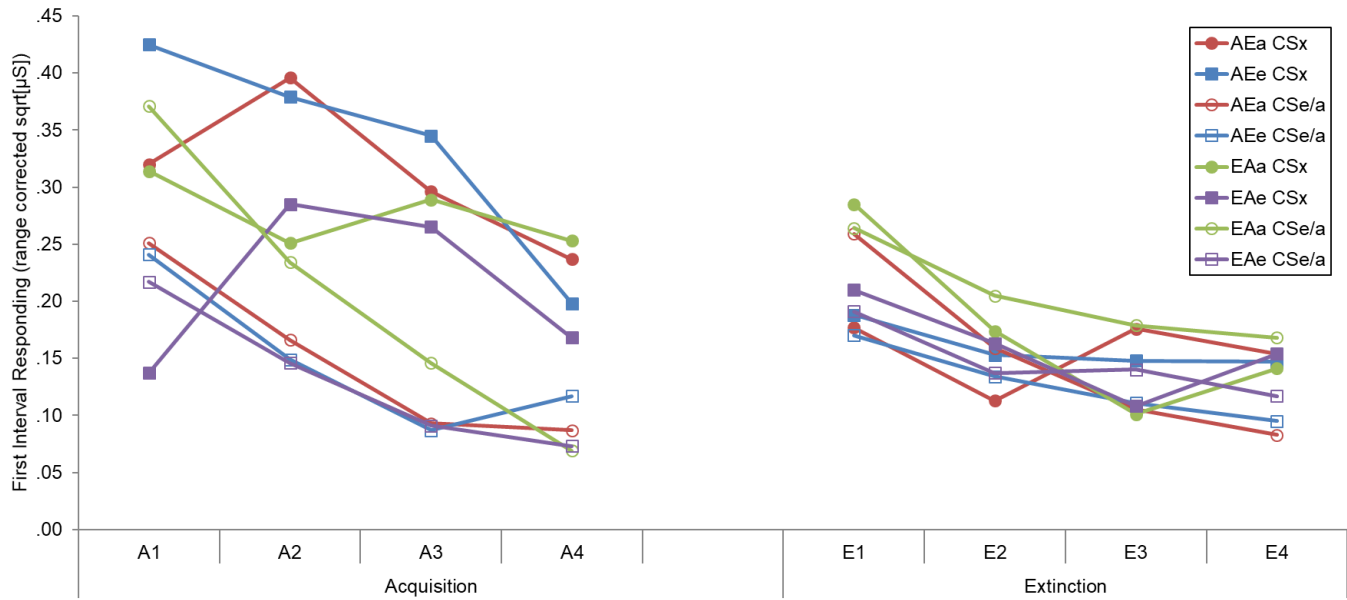


Figure 1. First interval electrodermal responding recorded during acquisition and extinction for the different groups. During acquisition, the CSa is presented and during extinction the CSe is presented.

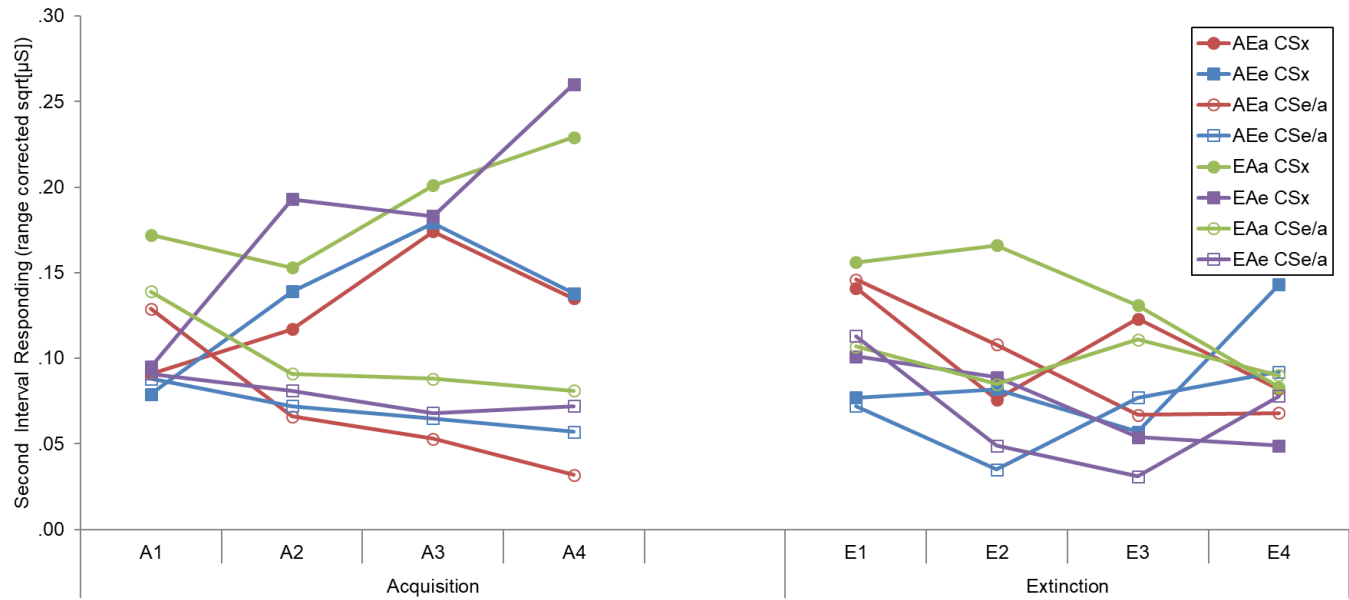


Figure 2. Second interval electrodermal responding recorded during acquisition and extinction for the different groups. During acquisition, the CSa is presented and during extinction the CSe is presented.

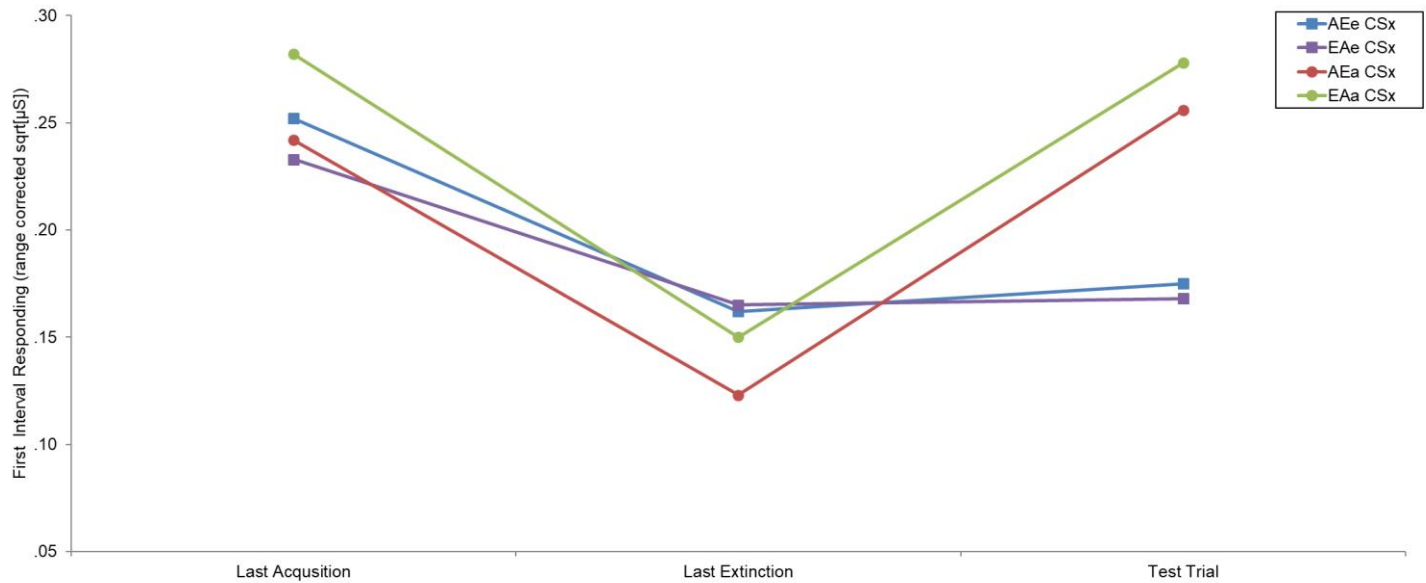


Figure 3. First interval electrodermal responding recorded to CSx during the last trial of acquisition, the last trial of extinction, and the test trial for the different groups. For AEe and AEa groups comparisons should be made from extinction to test. For EAe and EAa groups comparisons should be made from acquisition to test.

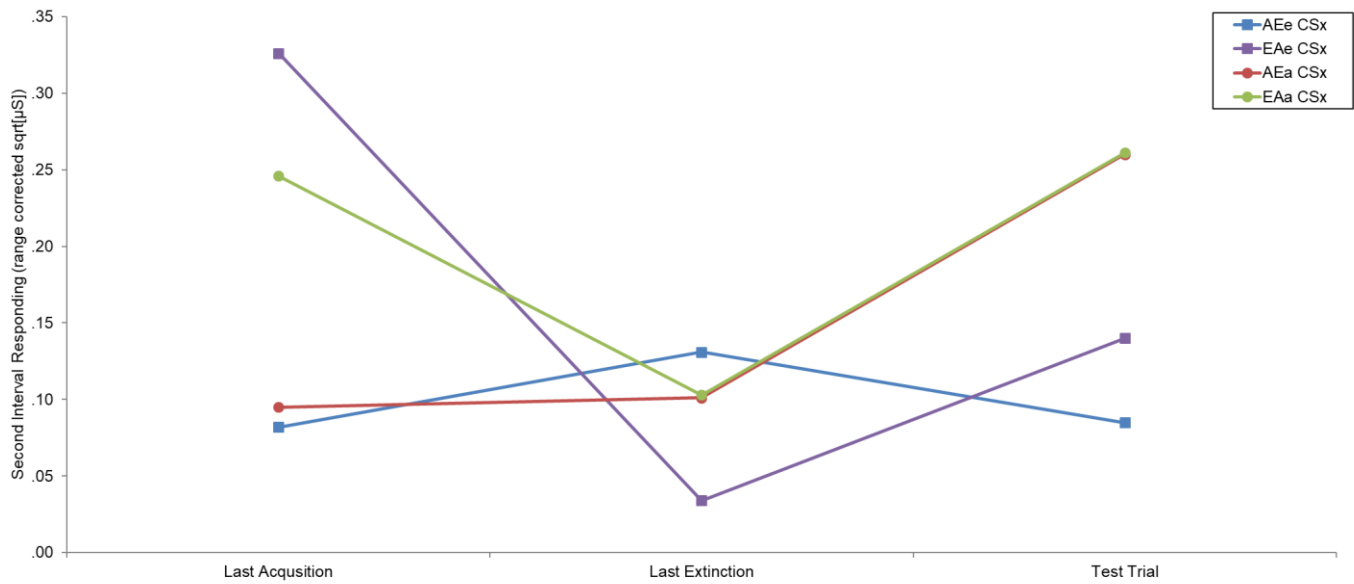


Figure 4. Second interval electrodermal responding recorded to CSx during the last trial of acquisition, the last trial of extinction, and the test trial for the different groups. For AEe and AEa groups comparisons should be made from extinction to test. For EAe and EAa groups comparisons should be made from acquisition to test.